62 | Cranial Nerve VII: The Facial Nerve and Taste

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Definition

The motor portion, or the facial nerve proper, supplies all the facial musculature. The principal muscles are the frontalis, orbicularis oculi, buccinator, orbicularis oris, platysma, the posterior belly of the digastric, and the stapedius muscle. In nuclear or infranuclear ("peripheral") lesions, there is a partial to complete facial paralysis with smoothing of the brow, open eye, flat nasolabial fold, and drooping of the mouth ipsilateral to the lesion. Supranuclear ("central") lesions spare the brow and eyelid musculature; there is flattening of the nasolabial fold and drooping of the mouth contralateral to the lesion.

The sensory portion, or intermediate nerve, has the following components: (1) taste to the anterior two-thirds of the tongue; (2) secretory and vasomotor fibers to the lacrimal gland, the mucous membranes of the nose and mouth, and the submandibular and sublingual salivary glands; (3) cutaneous sensory impulses from the external auditory meatus and region back of the ear. Abnormalities of taste include *ageusia* (lack of taste); *hypogeusia* (diminished taste acuity); *dysgeusia* (unpleasant, obnoxious, or perverted taste).

Technique

Motor

Careful and thoughtful observation is the key to discerning subtle signs of weakness of muscles supplied by the motor portion. Note especially the blink, nasolabial folds, and corners of the mouth. Asymmetry is the clue to unilateral weakness and is best perceived during conversation when the patient is unaware of being observed.

1. Blink: The eyelid on the affected side closes just a trace later than the opposite eyelid.
2. Nasolabial folds: The weak one is flatter.
3. Mouth: The affected side droops and participates manifestly less in speaking.

Ask the patient to look up or wrinkle the forehead; inspect for asymmetry. Ask him or her to close the eyes tightly. Look for incomplete closure or incomplete "burying" of the eyelashes on the affected side. Observe the nasolabial folds and mouth while the patient is concentrating on the eyes. As the orbicularis oculi contract tightly, there are milder associated contractions of muscles about the mouth and nose; these milder contractions are better suited to displaying slight weakness than when these muscles are tested directly.

Ask the patient to smile, show you teeth, or pull back the corners of the mouth. Look for asymmetry about the mouth.

The most subtle signs of mild facial weakness are the blink reflex and incomplete lid closure. Observe the blink reflex during conversation, or tap gently on the glabella with your index finger or reflex hammer in an attempt to bring out a mild asymmetry of blink. If you strongly suspect but are having difficulty confirming a mild facial weakness, ask the patient to lie flat on the examining table with face up. Slide the patient's head off the examining table so the head is below the body. This forces the eyelids to work against gravity. Now ask the patient to close both eyes and inspect for incomplete closure. Tap on the glabella and note asymmetry of blink.

The facial nerve participates in a number of reflexes (DeJong, 1979). Assessment of these reflexes provides valuable additional information about facial nerve function. Table 62.1 lists these reflexes, the method of eliciting them, and their clinical interpretation. Some of these reflexes are discussed in more detail in Chapter 71 (Suck, Snout, Palmomental, and Grasp Reflexes).

Taste

The four primary tastes are bitter, sweet, sour, and salty. Screen for disorders of sweet or salty taste with salt and sugar. With the patient's eyes closed and tongue protruded, take a tongue blade and smear a small amount of salt or sugar on the lateral surface and side of the tongue. Instruct the patient to tell you the identity of the substance. Rinse the mouth thoroughly and repeat the test on the other side, using a different substance.

Basic Science

The cortical fibers of the facial nerve proper originate from the lower third of the motor strip. They course in the genu of the internal capsule and the middle third of the cerebral peduncle, supplying the seventh nucleus in the lower pons. The supranuclear innervation is bilateral to the muscles of the forehead and eyes but only contralateral to the muscles of the lower part of the face. This accounts for the sparing of the upper facial muscles with a contralateral cortical lesion. Figure 62.1 shows the anatomy of the facial nerve.

The facial nucleus participates in the corneal reflex. Corneal pain and temperature fibers go through the ophthalmic division of the fifth cranial nerve to the spinal nucleus of the fifth and thence to the ipsilateral seventh nucleus, causing the eyelid to blink. There are also central connections between the facial nucleus and the nuclei or projection systems of the second, third, fourth, sixth, and eighth cranial nerves. These connections coordinate movements among the eyelids and eyeballs and set up certain reflexes such as the blink reflex on exposure to strong light or a loud sound. Table 62.1 lists these reflexes.

The motor fibers for the facial muscles exit from the motor nucleus, curl up and around the sixth nucleus, and descend laterally from the lower pons.
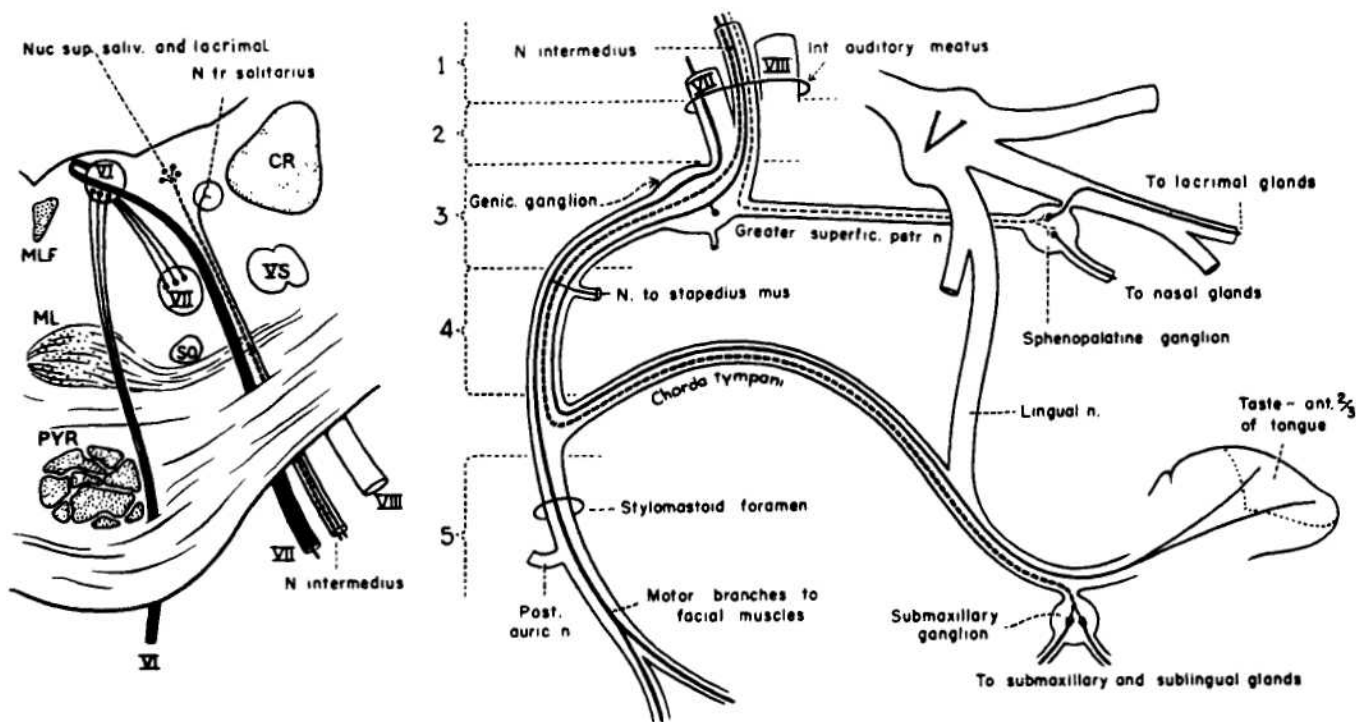
Figure 62.1
Origin and distribution of various components of the facial nerve. Symptomatology of lesions at various levels, 1 to 5, of this nerve is listed in Table 62.4. The facial, intermedius, and acoustic nerves illustrated to the left continue in the drawing on the right. CR, corpus resiforme; MLF, medial longitudinal fasciculus; ML, medial lemniscus, coursing vertically through corpus trapezoideum; PYR, pyramidal bundles in pars basilaris pontis; SO, superior olivary nucleus; VS, nucleus of spinal tract of the fifth nerve. *From* Haymaker W, Kuhlenbeck H. Disorders of the brainstem and its cranial nerves. In: Baker AB, Joynt RF, eds. Clinical neurology. Philadelphia: Lippincott, 1979;3:chap. 40. By permission.

The intermediate nerve joins the motor segment at the point where it exits from the pons. The intermediate nerve is composed of contributions from three areas:

1. The superior salivary nucleus in the pons supplies secretory fibers. They go to (a) the lacrimal, nasal, and palatine glands (via the greater superficial petrosal nerve) and (b) the submandibular and sublingual salivary glands (via the chorda tympani nerve).
2. The gustatory (solitary) nucleus in the medulla supplies sensory fibers. These fibers go to taste buds on the anterior two-thirds of the tongue (via the chorda tympani nerve).
3. The dorsal part of the trigeminal tract supplies fibers that convey cutaneous sensation from the external auditory meatus and skin behind the ear (distributed with the facial nerve proper).

First-order neurons are in the geniculate ganglion. Table 62.2 summarizes the brainstem nuclei of the facial and intermediate nerves.

The facial nerve proper and intermediate nerve lie in the cerebellopontine angle with the sixth and eighth cranial nerves. The seventh, intermediate, and eighth nerves enter the internal auditory meatus. The facial and intermediate nerves then enter the facial canal of the petrous portion of the temporal bone. The geniculate ganglion of the intermediate nerve is in this canal. The greater superficial petrosal nerve is destined for the lacrimal, nasal, and palatine glands. It leaves just distal to the geniculate ganglion. The nerve to the stapedius muscle is given off next. The facial

and intermediate nerves then descend to the stylomastoid foramen, giving off the chorda tympani at either the stylomastoid foramen or varying distances proximal to it. The chorda tympani supplies the anterior two-thirds of the tongue and the submandibular and sublingual glands. The motor part of the facial nerve leaves the stylomastoid foramen and supplies the facial musculature. A major part of the nerve forms a plexus within the parotid gland. Table 62.3 lists the branches of the facial nerve, beginning centrally and proceeding distally.

Taste sensibility is composed of four qualities: sweet, salt, sour, and bitter. Taste receptors are located on the tongue, palate, pharynx, and larynx. Although all four qualities can be perceived throughout, there is considerable localization. The tongue, especially the tip and edges, is most sensitive to sweet and salt; the palate, to sour and bitter. The receptors are taste buds. Up to 8 are on each of the fungiform papillae on the anterior two-thirds of the tongue, and up to 100 on each of the circumvallate papillae on the posterior part of the tongue. The taste buds are barrel shaped with a pore opening. Chemoreceptive taste hairs project into the barrel from neuroepithelial sensory cells. Impulses from these cells are transmitted to the brainstem. Afferent fibers from the anterior two-thirds of the tongue travel via the lingual nerve to the chorda tympani and then as described above to the gustatory nucleus. Taste fibers from the posterior third of the tongue, the palate, and the palatal arches travel via the glossopharyngeal nerve and the nodosal ganglion, also ending in the gustatory nucleus. There are two ascending pathways from the gustatory nucleus. One goes to the hypothalamus. The other goes to the thalamus and

Table 62.1
Reflexes Involving the Facial Nerve

Orbicularis oculi reflex
Percussion causes reflex contraction of the eye muscle. The reflex is known as the supraorbital, glabellar, or nasopalpebral reflex, depending upon the site of the stimulus. Both eyes usually close, with the contralateral response being weaker. The trigeminal nerve is the afferent side and the facial nerve the efferent side of the reflex. Light and sound can also produce the reflex, with the optic and acoustic nerves providing the afferent side.

The response is weak or abolished in nuclear and peripheral lesions, and present or exaggerated in supranuclear lesions. It is exaggerated in Parkinsonism and cannot be voluntarily inhibited.

Palpebral-oculogyric reflex
The eyeballs deviate upward when the eyes are closed, both when awake and asleep. The afferent arc is proprioceptive impulses carried through the facial nerve to the medial longitudinal fasciculus. The oculomotor nerve to the superior rectus muscles forms the efferent side.

In peripheral and nuclear lesions an exaggeration of this reflex is known as Bell's phenomenon.

Orbicularis oris reflex
Percussion on the side of the nose or the upper lip causes ipsilateral elevation of the angle of the mouth and upper lip. The reflex arc is composed of the fifth and seventh nerves. Synonyms: nasomental, buccal, oral, or perioral reflex.

This reflex disappears after about the first year of life, recurring with supranuclear facial nerve lesions and with extrapyramidal diseases, such as parkinsonism.

Snout reflex
Tapping the upper lip lightly with a reflex hammer, tongue blade, or finger causes bilateral contraction of the muscles around the mouth and base of the nose. The mouth resembles a snout.

This is an exaggeration of the orbicularis oris reflex. It is present with bilateral supranuclear lesions and in diffuse cerebral diseases, such as various causes of dementia.

Suck reflex
Sucking movements of lips, tongue, and mouth are brought about by lightly touching or tapping on the lips. At times merely bringing an object near the lips produces the reflex.

Occurs in patients with diffuse cerebral lesions. The snout reflex occurs in similar circumstances.

Palmomental reflex
A stimulus of the thenar area of the hand causes a reflex contraction ipsilaterally of the orbicularis oris and mentalis muscles.

A number of normal individuals have this reflex, and also patients with diffuse cerebral disease. It is significant when other similar reflexes are also present.

Corneal reflex
Stimulation of the cornea with a wisp of cotton produces reflex closure of both ipsilateral (strongest) and contralateral eyelids. The fifth nerve carries the afferent impulses, and the facial nerve the efferent impulses.

Table 62.2
Brainstem Nuclei and Functions of the Facial Nerve

Brainstem structure	Functions and comments
Motor	
Facial nucleus	Motor to facial muscles: frontalis, orbicularis oculi, platysma, buccinator, posterior belly of digastric, stapedius, stylohyoid, soft palate. Brachial motor nucleus.
Lacrimal nucleus	Visceral motor (parasympathetic) to lacrimal gland for tear secretion.
Superior salivatory nucleus	Visceral motor (parasympathetic) to salivary glands for saliva secretion; also nasal glands.
Sensory	
Nucleus solitarus	Special visceral sensory. From taste buds of anterior two-thirds of tongue. Geniculate ganglion.
Nucleus of spinal tract and main sensory nucleus V	General somatic sensory. Sensation from anterior nasopharynx, including upper part of hard and soft palate. Also from tympanic membrane, part of external auditory canal, lateral surface of ear, and area behind the ear and over the mastoid process.

Table 62.3
The Branches of the Facial Nerve Beginning Centrally

Branch	Comments
Greater superficial petrosal nerve	Leaves just distal to geniculate ganglion, which lies in facial canal of the petrous portion of the temporal bone. Supplies lacrimal, nasal, and palatine glands.
Nerve to stapedius muscle	Next branch to be given off, still within facial canal.
Chorda tympani	Leaves about 5 mm or less before the stylomastoid foramen. Taste to anterior two-thirds of tongue, submaxillary and sublingual salivary glands.
Motor branches	At exit from stylomastoid foramen the posterior auricular, digastric, and stylohoid branches are given off. In the parotid gland the nerve divides into two branches, the temporofacial and cervicofacial, which supply the remaining muscles.

then to the gustatory center of the cortex, which is probably area 43 in the parietal operculum.

Clinical Significance

Motor

A lesion involving the nuclear or infranuclear portion of the facial nerve will produce a peripheral facial palsy. If all motor components are involved, there is complete paralysis of all facial muscles on the involved side. The brow is smooth, the eye does not close, the nasolabial fold is flat, and that side of the mouth droops. There is no movement at all. The paradigm of this type of involvement is Bell's palsy. Idiopathic Bell's palsy may strike at any age, often after a mild viral illness. Recovery is over a period of weeks to months and is variable. The cause of the idiopathic variety is unknown. Sequelae to Bell's palsy include the following:

1. Interfacial synkinesis: When the eyes close, the mouth will twitch. This occurs when the regenerating nerve fibers do not grow back into the proper muscles. The synkinetic movements are almost always present on the involved side.
2. Because of the contractures, the face at rest may be more deeply etched on the side of the previous palsy. This can give a false impression of weakness on the opposite side.

Other causes of peripheral seventh nerve palsy include: neoplasm, trauma, middle ear infections, parotid gland surgery, granulomatous or carcinomatous meningitis, and diabetes. The disturbances of function produced by these lesions need not be complete. An important clinical point is that the clinical manifestations of these disorders are indistinguishable from idiopathic Bell's palsy.

Supranuclear involvement produces contralateral paralysis of the lower facial muscles and sparing of the upper muscles due to the bilateral supranuclear innervation of the latter. Subtle weakness is often difficult to confirm. Many, perhaps even most, normal individuals have mild lower facial asymmetries, making interpretation difficult.

Anatomic localization of lesions is made by the characteristics of the dysfunction and associated structures involved. Table 62.4 gives the location of lesions and the clinical manifestations. Refer to Figure 62.1 to see exactly where the lesions occur.

Taste

Patients with Addison's disease, pituitary insufficiency, or cystic fibrosis have an increased ability to detect the four primary tastes. Taste acuity returns to normal with glucocorticoid therapy in the cases of adrenal hypofunction. Conversely, penicillamine therapy may be associated with a decreased acuity for the four primary tastes. A wide variety of conditions may cause decreased or absent taste. They are listed in Table 62.5.

Gustatory sweating is sweating on the face associated eating. It is seen in the following circumstances:

1. Diabetes mellitus, presumably due to the diabetic autonomic neuropathy
2. Frey's syndrome: gustatory sweating after injury, infection, or surgery of the parotid gland
3. After upper dorsal sympathectomy
4. Physiological, occurring after eating highly spiced food.

Table 62.4
Localization of Facial Nerve Lesions

Lesion location	Manifestations
Above the facial nucleus (supranuclear lesion)	Contralateral paralysis of lower facial muscles with relative preservation of upper muscles. Lesion located either in brainstem or cortex.
Pons (nuclear or fascicular lesion)	Ventral pontine lesion (of Millard-Gubler): ipsilateral facial monoplegia, lateral rectus palsy (VI), contralateral hemiplegia (corticospinal fibers). Pontine tegmentum lesion (of Foville): ipsilateral facial monoplegia; contralateral hemiplegia (corticospinal fibers); paralysis of conjugate gaze to side of lesion (pontine paramedian reticular formation).
Cerebellopontine angle (peripheral nerve lesion). 1 in Figure 62.1	Ipsilateral facial monoplegia, loss of taste to anterior two-thirds of tongue, impairment of salivary and tear secretion, hyperacusis (if VIII is not affected). Additional cranial nerves may be involved: deafness, tinnitus, vertigo (VII): sensory loss over face and absence of corneal reflex (V); ipsilateral ataxia (cerebellar peduncle).
Facial canal between internal auditory meatus and geniculate ganglion (peripheral nerve type lesion here and subsequently). 2 in Figure 62.1	Same as above except cranial nerves other than VII are not involved.
Facial canal between geniculate ganglion and nerve to stapedius muscle. 3 in Figure 62.1	Facial monoplegia; impaired salivary secretion; loss of taste; hyperacusis.
Facial canal between nerve to stapedius and leaving of chorda tympani. 4 in Figure 62.1	Facial monoplegia; impaired salivary secretion; loss of taste.
After branching of chorda tympani. 5 in Figure 62.1	Facial paralysis, distribution related to site of lesion.

Numbers refer to Figure 62.1.

Table 62.5
Examples of Disorders Reported to Be Associated with Gustatory Dysfunction

Endocrine
  Adrenal cortical insufficiency
  Congenital adrenal hyperplasia
  Panhypopituitarism
  Cushing's syndrome
  Hypothyroidism
  Diabetes mellitus
  Turner's syndrome
  Pseudohypoparathyroidism
Inflammatory
  Infections:
    Candida
    Gingivitis
    Herpes simplex
    Periodontitis
    Sialadenitis
  Autoimmune:
    Pemphigus
    Sjøgren's syndrome
Local alterations of taste buds or papillae
  Chemicals, drugs
  Xerostomia
Neurologic
  Bell's palsy
  Familial dysautonomia
  Head trauma
  Middle ear operations with Cretinism manipulation or damage to chorda tympani
  Multiple sclerosis
  Raeder's paratrigeminal syndrome
Nutritional
  Cachexia
  Chronic renal failure
  Cirrhosis of the liver
  Niacin (vitamin B_3 deficiency)
  Zinc deficiency
Psychiatric
  Depression
  Schizophrenia
Tumors
  Oral cavity cancer
  Base of skull neoplasia

Source: From Doty RL, Kimmelman CP. Smell and taste and their disorders. In: Asbury AK, McKhann GM, McDonald WI, eds. Diseases of the nervous system. Philadelphia: W.B. Saunders 1986;1:466–78.

No proposed mechanism explains the occurrence of this phenomenon in all cases.

References

Brodal A. Neurological anatomy in relation to clinical medicine. 3rd ed. New York: Oxford University Press, 1981.

DeJong RN. The neurologic examination. 4th ed. New York: Harper & Row, 1979;178–98.

Doty RL. A review of olfactory dysfunctions in man. Am J Otolaryngol 1979;1:57–79.

Doty RL, Kimmelman CP. Smell and taste and their disorders. In: Asbury AK, McKhann GM, McDonald WI, eds. Diseases of the nervous system. Philadelphia: W.B. Saunders, 1986;1:466–78.

Haymaker W, Kuhlenbeck H. Disorders of the brainstem and its cranial nerves. In: Baker AB, Joynt RF, eds. Clinical neurology. Philadelphia: Lippincott, 1985;3:chap. 40.

Karnes WE. Diseases of the seventh cranial nerve. In: Dyck PJ, Thomas PK, Lambert EH, Bunge R, eds. Peripheral neuropathy. 2nd ed. Philadelphia: W.B. Saunders, 1984;2:1266–99.

Schiffman SS. Taste and smell in disease. N Engl J Med 1983; 308:1275–79, 1337–43.